The orbitofrontal cortex (OFC) function is integral to cue-guided behaviour based on the current, up-to-the-moment expected value of predicted rewards [REFS Walton review]. Thus, following OFC damage, rodents show a profound inability to appropriately inhibit responding to predictive cues when the expected reward is no longer valuable (e.g. outcome devaluation procedures; REFS) or omitted as in extinction procedures [REFS]. Findings such as these have historically been interpreted as evidence for the function of the OFC in inhibitory control over “prepotent” responses (Murray, O’Doherty, & Schoenbaum, 2007). This general inhibition hypothesis is usually dismissed based on a number of findings that OFC lesions do not disrupt the ability to inhibit some behaviours. For example, OFC lesions do not impair monkeys from learning to inhibit an innate preference for selecting larger food rewards (Chudasama, Kralik, & Murray, 2007). Similarly, OFC lesions do not impair rats from learning to inhibit an established response in a go, no-go discrimination task (Schoenbaum, Setlow, Nugent, Saddoris, & Gallagher, 2003). Instead, the deficits reported in outcome devaluation, reversal and extinction studies [REFS] following OFC lesions have can be parsimoniously explained by the OFC encoding the current expected value of predicted outcomes.

Recently, (Panayi & Killcross, 2014) directly tested the role of rodent lateral OFC in the representation of outcome expectancy information that may drive prediction error based learning. After learning that a cue reliably predicted the delivery of food reward across many training sessions, OFC function was then temporarily disrupted by infusions of muscimol during sessions in which the food was no longer delivered following the cue i.e. Pavlovian extinction. Extinction learning should generate large prediction errors caused by the discrepancy between the expected delivery of the reward and its omission. If disrupting OFC function suppresses expected value signals, then prediction error signalling should be blocked in extinction and rats should failed to learn that the cue no longer predicts reward. In line with this prediction, (Panayi & Killcross, 2014) reported that extinction learning was disrupted between sessions following OFC inactivation.

A key initial behavioural finding following lesion damage to the OFC in primates was impaired suppression of responding in an extinction procedure in which previously rewarded cues were no longer rewarded [REFS Butter]. The OFC has traditionally been hypothesised as a prefrontal locus of behavioural inhibition [REFS], however recent theories of orbitofrontal function propose that the OFC is necessary for modifying behaviour based on the current up-to-the-moment expected value of outcomes [REFS]. For example, after learning that a cue reliably predicts food reward, if the food is subsequently paired with illness to render it aversive, rats with excitotoxic lesions of the OFC will abnormally persist in responding in anticipation of the now devalued outcome predicted by the cue [REFS]. Conversely, in Pavlovian over-expectation procedures a functional OFC is required to selectively increase responding when two predictive cues are compounded to predict an increase in expected reward value (Takahashi et al., 2009). Thus, OFC function is critical to both increasing and decreasing behaviour appropriately when outcome values change.

While many authors have suggested that the role of the OFC is not merely the inhibition of inappropriate responding, it is not clear whether the OFC is directly involved in the acquisition of conditioned inhibition